

# EXHIBIT 1

## DECLARATION OF RANDY SCOTT, Ph.D. UNDER 37 C.F.R. § 1.132

I, Randy Scott, Ph.D. declare and say as follows:

1. I hold a Bachelor of Science degree in Chemistry from Emporia State University and a Ph.D. in Biochemistry from the University of Kansas.
2. I am Chairman and Chief Executive Officer of Genomic Health, Inc., a life science company founded in August of 2000 located in Redwood City, California, conducting sophisticated genomic research to develop clinically validated molecular diagnostics, which provide individualized information on the likelihood of disease recurrence and response to certain types of therapy.
3. In 1991, I co-founded Incyte Pharmaceuticals, Inc., the world's first genomic information business. I served the company in multiple capacities, including Chairman of the Board from August 2000 to December 2001, President from January 1997 to August 2000, and Chief Scientific Officer from March 1995 to August 2000. Under my leadership, Incyte has created the LifeSeq Gold<sup>®</sup> gene sequence and expression database, an industry standard and the most comprehensive collection of biological information in the world. I have also led Incyte to expand its focus beyond gene sequence databases to include the research and application of gene expression, SNPs (single nucleotide polymorphisms), and proteomics.
4. I am an inventor on several issued patents, and authored over 40 scientific publications in the fields of protein biology, gene discovery, and cancer.
5. My Curriculum Vitae is attached to and serves part of this Declaration.
6. All statements made in this Declaration are based on my more than 15 years of personal experience with the DNA microarray technique and its various uses in the diagnostic and therapeutic fields, and my familiarity with the relevant art.
7. The DNA microarray technology is based on hybridizing arrayed nucleic acid probes of known identity with target nucleic acid to determine the identity and/or expression levels (abundance) of target genes. DNA microarrays work by exploiting the ability of a given

mRNA molecule to hybridize to the DNA template from which it originated. By using an array containing many DNA samples, scientists can determine, in a single experiment, the expression levels of hundreds or thousands of genes within a sample by measuring the amount of mRNA bound to each site on the array. The amount of mRNA bound to the spots on the microarray is precisely measured, generating a profile of gene expression in the sample.

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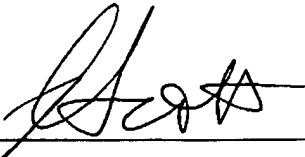
8. DNA microarray analysis has been extensively used in drug development and in diagnosis of various diseases. For instance, if a certain gene is over-expressed in a particular form of cancer relative to normal tissue, researchers use microarray chips to determine whether a drug candidate will reduce over-expression, and thereby cause cancer remission. In addition, if a gene has been identified to be over-expressed in a certain disease, such as a certain type of cancer, it can be used to diagnose that disease. Due to its importance in drug discovery and in the field of diagnostics, microarray technology has not only become a laboratory mainstay but also created a world-wide market of over \$600 million in the year of 2005. A long line of companies, including Incyte, Affymetix, Agilent, Applied Biosystems, and Amersham Biosciences, made microarray technology a core of their business.

9. Correlation between mRNA and protein levels can be assessed by a variety of methods suitable for measuring protein expression levels, including, for example, SDS-polyacrylamide gel electrophoresis (SDS-PAGE), two-dimensional fluorescence-difference gel electrophoresis (DIGE), mass spectrometric approaches, microsequencing, and a combination of these and similar known techniques, however, direct measurement of protein expression levels remains non-trivial.

10. One reason for the success and wide-spread use of the DNA microarray technique, which has led to the emergence of a new industry, is that generally there is a good correlation between mRNA levels determined by microarray analysis and expression levels of the translated protein. Although there are some exceptions on an individual gene basis, it has been a consensus in the scientific community that elevated mRNA levels are good predictors of increased abundance of the corresponding translated proteins in a particular tissue. Therefore, diagnostic markers and drug candidates can be readily and efficiently screened and identified using this technique, without the need to directly measure individual protein expression levels.

11. I further declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the Patent.

Date: August 11, 2006

  
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Randy Scott, Ph.D.

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8/11/06 11:00 AM (39766.7000)

Randy W. Scott, Ph.D.  
Genomic Health  
301 Penobscot  
Redwood City, CA 94022

**EDUCATION:**

1979 B.S., Chemistry, Emporia State University, Emporia Kansas  
1983 Ph.D., Biochemistry, University of Kansas, Lawrence Kansas

**WORK EXPERIENCE:**

**2000-present GENOMIC HEALTH, INC., Cofounder**

- Chairman & CEO, (2000-present)  
Founded a new genomics company and raised over \$100 million to bring personalized medicine to clinical practice. Selected by Red Herring Magazine as one of the Top 100 private technology companies in North America in 2005

**1991-2000 INCYTE, Cofounder**

- Chairman of the Board (2000-2001)  
Helped lead the transition to a new management team and transition to drug development
- President and Chief Scientific Officer (1997-2000)  
Responsible for Research & Development, Operations, Marketing & Sales. Built the world's first genomic information business with peak sales of over \$200 million per year including 19 out of the worlds top 20 pharmaceutical companies as subscribers
- Vice President and Chief Scientific Officer (1991-1997)  
Built recombinant DNA therapeutic product portfolio and led the launch of the genomics business

**1985-91 INVITRON CORPORATION**

- Sr. Director of Research (1998-1991)  
Responsible for Research & Development.
- Director of Protein Biochemistry (1985-1988)  
Responsible for building the protein purification group for a cGMP manufacturing facility producing recombinant proteins, including monoclonal antibodies, tPA and Factor VIII.

**1983-85 UNIGENE LABORATORIES, Fairfield, New Jersey**

- Sr. Scientist, Dept. of Protein Biochemistry  
Led effort to work on IgA proteases linked to meningococcal infections

**OTHER EXPERIENCE:**

**2005- Present AMERICAN CLINICAL LABORATORY ASSOCIATION**

- Member, Board of Directors

**1997-2000 DIADEXUS, INC., Cofounder**

- Member, Board of Directors, (1997-2000)  
Worked with George Poste (CSO, SmithKline, Beecham) to establish a diagnostics joint venture between Incyte and SmithKline

**Awards:**

2001 Genome Technology Magazine 2001 All-Star  
1999 Forbes Magazine list of Biotech's Top 25 Influential Insiders  
1997 Ernst & Young/NASDAQ Silicon Valley Entrepreneur of the Year for Life Sciences  
1987 Small Business Innovation Research Grant Award (Principal Investigator): "Azurophil-Derived Bactericidal Factor" Grant # SSS-5 (K) 1R43AI24409-011987  
1983 Phillip Newmark Research Award, University of Kansas, 1983  
1982 Borgendale Graduate Seminar Award, University of Kansas.

**Publications:**

Low, D.A., Cunningham, D.D., Scott, R.W., and Baker, J.B., "Interactions of Serine Proteases with Human Fibroblasts: Regulation by Protease Nexin, A Cellular Component with Similarities to Antithrombin III." in Receptor-Mediated Binding and Internalization of Toxins and Hormones (Middlebrook, J.L. and Kohn, L.S. eds.) pp. 259-270, Academic Press, New York (1982).

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- Scott, R.W.**, "Purification, Characterization, and Functional Studies of Protease Nexin." Ph.D. Thesis, University of Kansas (1983).
- Scott, R.W.**, Eaton, D.L., Duran, N. and Baker, J.B. Regulation of Extracellular Plasminogen Activator by Human Fibroblasts. The Role of Protease Nexin. J. Biol. Chem. 258, 4397-4403 (1983).
- Scott, R.W.**, and Baker, J.B., Purification of Human Protease Nexin. J. Biol. Chem. 258, 10439-10444 (1983).
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- McGrogan, M., Kennedy, J., Li, M.P., Hsu, C., **Scott, R.W.**, Simonsen, C.C., and Baker, J.B., Molecular Cloning and Expression of Two Forms of Human Protease Nexin I, Bio/Technology 6: 172 (1988).
- Otsuka FL, Cance WG, Dilley WG, **Scott RW**, Davie JM, Welch MJ, Wells SA Jr., A Potential New Radiopharmaceutical for Parathyroid Imaging: Radiolabeled Parathyroid-specific Monoclonal Antibody -I. Evaluation of 125-I-labeled Antibody in a Nude Mouse Model System. Int. J. Rad. Appl. Instrum. B. 15:305-11, 1988
- Otsuka FL, Cance WG, Dilley WG, **Scott RW**, Davie JM, Wells SA Jr., Welch MJ A Potential New Radiopharmaceutical for Parathyroid Imaging: Radiolabeled Parathyroid-specific Monoclonal Antibody -II. Comparison of 125-I and 111-In-labeled Antibodies. Int. J. Rad. Appl. Instrum. B. 15:305-11, 1988
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- Rogy MA, Oldenburg HS, Calvano SE, Montegut WJ, Stackpole SA, Van Zee KJ, Marra MN, **Scott RW**, Seilhammer JJ, Moldawer LL. The Role of Bactericidal/Permeability-Increasing Protein in the Treatment of Primate Bacteremia and Septic Shock. *J Clin. Immunol.* 14: 120-33, 1994
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- Wilde, G.G., Seilhamer, J.J., McGrogan, M., Ashton, N., Snable, J.L., Lane JC, Leong, SR, Thornton, MB, Miller, KL, **Scott RW**, and Marra, MN "Bactericidal/Permeability-Increasing Protein and Lipopolysaccharide (LPS)-Binding Protein: LPS Binding Properties and Effects on LPS-Mediated Cell Activation" *J. Biol. Chem.* 269:17411-17416, 1994
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**Scott RW**, Gene Patents and Other Genomic Inventions. Published Hearing before the Subcommittee on Courts and Intellectual Property of the Committee on the Judiciary House of Representatives, One Hundred Sixth Congress, Second Session, July 13, 2000 Serial No. 121. pp. 44-55 . U.S. Government Printing Office Washington, 2000

**Issued Patents:**

U.S. Patent # 4,898,826 Issued Feb. 6, 1990  
A Method for Solubilization of Tissue-Type Plasminogen Activator.

U.S. Patent # 5,006,252 Issued April 9, 1991  
Recombinant Purified Protease Nexin.

U.S. Patent #5,032,574 Issued July 16, 1991  
Novel Antimicrobial Peptide, Compositions Containing Same and Uses Thereof.

U.S. Patent #5,087,368 Issued Feb. 11, 1992  
Purified Protease Nexin

U.S. Patent #5,089,274 Issued Feb. 18, 1992  
Use of Bactericidal/Permeability Increasing Protein or Biologically Active Analogs Thereof to Treat Endotoxin-Related Disorders

U.S. Patent #5,112,608 Issued May 12, 1992  
Use of Protease Nexin-1 to Mediate Wound Healing

U.S. Patent #5,171,739 Issued December 15, 1992  
Treatment of Endotoxin-Associated Shock and Prevention Thereof Using a BPI Protein

U.S. Patent #5,187,089 Issued Feb. 16, 1993  
Protease Nexin-1 Variants Which Inhibit Elastase

U.S. Patent #5,196,196 Issued March 23, 1993  
Use of Protease Nexin-1 in Wound Dressings

U.S. Patent #5,206,017 Issued Apr. 27, 1993  
Use of Protease Nexin-1 as an Anti-inflammatory

U.S. Patent #5,210,027 Issued May 11, 1993  
DNA Encoding Novel Antimicrobial Polypeptide and Methods for Obtaining Such Polypeptide

U.S. Patent #5,278,049 Issued January 11, 1994  
Recombinant Molecule encoding Human Protease Nexin

U.S. Patent #5,234,912 Issued August 10, 1993  
Pharmaceutical Compositions Comprising Recombinant BPI Proteins and a Lipid Carrier and Uses Thereof

U.S. Patent #5,278,049 Issued January 11, 1994  
Recombinant Molecule encoding Human Protease Nexin

U.S. Patent #5,308,834 Issued May 3, 1994  
Treatment of Endotoxin-Associated Shock and Prevention Thereof Using BPI Protein

U.S. Patent #5,326,562 Issued July 5, 1994  
Pharmaceutical Dosage Unit for Treating Inflammation Comprising Protease Nexin-I

U.S. Patent #5,234,912 Issued August 10, 1993  
Pharmaceutical Compositions Comprising Recombinant BPI Proteins and a Lipid Carrier and Uses

U.S. Patent #5,278,049 Issued January 11, 1994  
Recombinant Molecule Encoding Human Protease Nexin

U.S. Patent #5,326,562 Issued July 5, 1994  
Pharmaceutical Dosage Unit for Treating Inflammation Comprising Protease Nexin-1

U.S. Patent #5,334,584 Issued August 2, 1994

## Recombinant, Non-Glycosylated BPI Protein and Uses Thereof

U.S. Patent #5,457,090 Issued October 10, 1995  
Protease Nexin-I Variants

U.S. Patent #5,470,825 Issued November 28, 1995  
Basophil Granule Proteins

U.S. Patent #5,476,839 Issued December 19, 1995  
Basophil Granule Proteins

U.S. Patent #5,495,001 Issued February 27, 1996  
Recombinant Purified Protease Nexin

U.S. Patent #5,747,283 Issued May 5, 1998  
Basophil Granule Proteins

U.S. Patent #5,770,694 Issued June 23, 1998  
Genetically Engineered BPI Variant Proteins

U.S. Patent #5,840,484 Issued November 24, 1998  
Comparative Gene Transcript Analysis

U.S. Patent #6,114,114 Issued September 5, 2000  
Comparative Gene Transcript Analysis

U.S. Patent #6,093,801 Issued July 25, 2000  
Recombinant Analogs of Bactericidal/Permeability Increasing Protein

U.S. Patent #6,160,104 Issued December 12, 2000  
Markers for Peroxisomal Proliferators

U.S. Patent #6,160,105 Issued December 12, 2000  
Monitoring Toxicological Responses

U.S. Patent #6,265,187 Issued July 24, 2001  
Recombinant Endotoxin Neutralizing Proteins

U.S. Patent #6,403,778 Issued June 11, 2002  
Toxicological Response Markers

U.S. Patent #6,372,431 Issued April 16, 2002  
Mammalian Toxicological Response Markers

U.S. Patent #6,553,317 Issued April 22, 2003  
Relational database and system for storing information relating to biomolecular sequences and reagents



## EXHIBIT 2

### SECOND DECLARATION OF PAUL POLAKIS, Ph.D.

I, Paul Polakis, Ph.D., declare and say as follows:

1. I am currently employed by Genentech, Inc. where my job title is Staff Scientist.
2. Since joining Genentech in 1999, one of my primary responsibilities has been leading Genentech's Tumor Antigen Project, which is a large research project with a primary focus on identifying tumor cell markers that find use as targets for both the diagnosis and treatment of cancer in humans.
3. As I stated in my previous Declaration dated May 7, 2004 (attached as Exhibit A), my laboratory has been employing a variety of techniques, including microarray analysis, to identify genes which are differentially expressed in human tumor tissue relative to normal human tissue. The primary purpose of this research is to identify proteins that are abundantly expressed on certain human tumor tissue(s) and that are either (i) not expressed, or (ii) expressed at detectably lower levels, on normal tissue(s).
4. In the course of our research using microarray analysis, we have identified approximately 200 gene transcripts that are present in human tumor tissue at significantly higher levels than in normal human tissue. To date, we have successfully generated antibodies that bind to 31 of the tumor antigen proteins expressed from these differentially expressed gene transcripts and have used these antibodies to quantitatively determine the level of production of these tumor antigen proteins in both human tumor tissue and normal tissue. We have then quantitatively compared the levels of mRNA and protein in both the tumor and normal tissues analyzed. The results of these analyses are attached herewith as Exhibit B. In Exhibit B, "+" means that the mRNA or protein was detectably overexpressed in the tumor tissue relative to normal tissue and "-" means that no detectable overexpression was observed in the tumor tissue relative to normal tissue.
5. As shown in Exhibit B, of the 31 genes identified as being detectably overexpressed in human tumor tissue as compared to normal human tissue at the mRNA level, 28 of them (i.e., greater than 90%) are also detectably overexpressed in human tumor tissue as compared to normal human tissue at the protein level. As such, in the cases where we have been able to quantitatively measure both (i) mRNA and (ii) protein levels in both (i) tumor tissue and (ii) normal tissue, we have observed that in the vast majority of cases, there is a very strong correlation between increases in mRNA expression and increases in the level of protein encoded by that mRNA.

6. Based upon my own experience accumulated in more than 20 years of research, including the data discussed in paragraphs 4-5 above and my knowledge of the relevant scientific literature, it is my considered scientific opinion that for human genes, an increased level of mRNA in a tumor tissue relative to a normal tissue more often than not correlates to a similar increase in abundance of the encoded protein in the tumor tissue relative to the normal tissue. In fact, it remains a generally accepted working assumption in molecular biology that increased mRNA levels are more often than not predictive of elevated levels of the encoded protein. In fact, an entire industry focusing on the research and development of therapeutic antibodies to treat a variety of human diseases, such as cancer, operates on this working assumption.
7. I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information or belief are believed to be true, and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful statements may jeopardize the validity of the application or any patent issued thereon.

Dated: 3-29-06

By: Paul Polakis

Paul Polakis, Ph.D.

DECLARATION OF PAUL POLAKIS, Ph.D.

I, Paul Polakis, Ph.D., declare and say as follows:

1. I was awarded a Ph.D. by the Department of Biochemistry of the Michigan State University in 1984. My scientific Curriculum Vitae is attached to and forms part of this Declaration (Exhibit A).
2. I am currently employed by Genentech, Inc. where my job title is Staff Scientist. Since joining Genentech in 1999, one of my primary responsibilities has been leading Genentech's Tumor Antigen Project, which is a large research project with a primary focus on identifying tumor cell markers that find use as targets for both the diagnosis and treatment of cancer in humans.
3. As part of the Tumor Antigen Project, my laboratory has been analyzing differential expression of various genes in tumor cells relative to normal cells. The purpose of this research is to identify proteins that are abundantly expressed on certain tumor cells and that are either (i) not expressed, or (ii) expressed at lower levels, on corresponding normal cells. We call such differentially expressed proteins "tumor antigen proteins". When such a tumor antigen protein is identified, one can produce an antibody that recognizes and binds to that protein. Such an antibody finds use in the diagnosis of human cancer and may ultimately serve as an effective therapeutic in the treatment of human cancer.
4. In the course of the research conducted by Genentech's Tumor Antigen Project, we have employed a variety of scientific techniques for detecting and studying differential gene expression in human tumor cells relative to normal cells, at genomic DNA, mRNA and protein levels. An important example of one such technique is the well known and widely used technique of microarray analysis which has proven to be extremely useful for the identification of mRNA molecules that are differentially expressed in one tissue or cell type relative to another. In the course of our research using microarray analysis, we have identified approximately 200 gene transcripts that are present in human tumor cells at significantly higher levels than in corresponding normal human cells. To date, we have generated antibodies that bind to about 30 of the tumor antigen proteins expressed from these differentially expressed gene transcripts and have used these antibodies to quantitatively determine the level of production of these tumor antigen proteins in both human cancer cells and corresponding normal cells. We have then compared the levels of mRNA and protein in both the tumor and normal cells analyzed.
5. From the mRNA and protein expression analyses described in paragraph 4 above, we have observed that there is a strong correlation between changes in the level of mRNA present in any particular cell type and the level of protein

expressed from that mRNA in that cell type. In approximately 80% of our observations we have found that increases in the level of a particular mRNA correlates with changes in the level of protein expressed from that mRNA when human tumor cells are compared with their corresponding normal cells.

6. Based upon my own experience accumulated in more than 20 years of research, including the data discussed in paragraphs 4 and 5 above and my knowledge of the relevant scientific literature, it is my considered scientific opinion that for human genes, an increased level of mRNA in a tumor cell relative to a normal cell typically correlates to a similar increase in abundance of the encoded protein in the tumor cell relative to the normal cell. In fact, it remains a central dogma in molecular biology that increased mRNA levels are predictive of corresponding increased levels of the encoded protein. While there have been published reports of genes for which such a correlation does not exist, it is my opinion that such reports are exceptions to the commonly understood general rule that increased mRNA levels are predictive of corresponding increased levels of the encoded protein.

7. I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information or belief are believed to be true, and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful statements may jeopardize the validity of the application or any patent issued thereon.

Dated: 5/07/04

By: Paul Polakis

Paul Polakis, Ph.D.

## CURRICULUM VITAE

PAUL G. POLAKIS  
Staff Scientist  
Genentech, Inc  
1 DNA Way, MS#40  
S. San Francisco, CA 94080

### EDUCATION:

Ph.D., Biochemistry, Department of Biochemistry,  
Michigan State University (1984)

B.S., Biology. College of Natural Science, Michigan State University (1977)

### PROFESSIONAL EXPERIENCE:

2002-present	Staff Scientist, Genentech, Inc S. San Francisco, CA
1999- 2002	Senior Scientist, Genentech, Inc., S. San Francisco, CA
1997 -1999	Research Director Onyx Pharmaceuticals, Richmond, CA
1992- 1996	Senior Scientist, Project Leader, Onyx Pharmaceuticals, Richmond, CA
1991-1992	Senior Scientist, Chiron Corporation, Emeryville, CA.
1989-1991	Scientist, Cetus Corporation, Emeryville CA.
1987-1989	Postdoctoral Research Associate, Genentech, Inc., South San Francisco, CA.
1985-1987	Postdoctoral Research Associate, Department of Medicine, Duke University Medical Center, Durham, NC

1984-1985

Assistant Professor, Department of Chemistry,  
Oberlin College, Oberlin, Ohio

1980-1984

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**EXHIBIT B**

	tumor mRNA	tumor IHC
UNQ2525	+	+
UNQ2378	+	+
UNQ972	+	-
UNQ97671	+	+
UNQ2964	+	+
UNQ323	+	+
UNQ1655	+	+
UNQ2333	+	+
UNQ9638	+	+
UNQ8209	+	+
UNQ6507	+	+
UNQ8196	+	+
UNQ9109	+	+
UNQ100	+	+
UNQ178	+	+
UNQ1477	+	+
UNQ1839	+	+
UNQ2079	+	+
UNQ8782	+	+
UNQ9646	+	-
UNQ111	+	+
UNQ3079	+	+
UNQ8175	+	+
UNQ9509	+	+
UNQ10978	+	-
UNQ2103	+	+
UNQ1563	+	+
UNQ16188	+	+
UNQ13589	+	+
UNQ1078	+	+
UNQ879	+	+